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Congenital Portosystemic Shunts in the Canine: A Case Report and Review of the Syndrome, Its Diagnosis and Treatment

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INTRODUCTION

Congenital anomalies of the portal vascular system which result in blood bypassing the hepatic parenchyma lead to the development of hepatic insufficiency. Most commonly seen in young dogs (mean age at onset of clinical signs in one report was 8.4 months[1]), cases of portosystemic shunts are characterized by a wide variety of clinical and pathologic abnormalities.

The portal vein is formed by the confluence of the splenic and cranial mesenteric veins at the level of the second lumbar vertebra.[2] It collects the blood from the stomach, intestines, pancreas and spleen. In all, two-thirds of the blood supply to the liver comes from the portal vein.[3]

The investigation of correlation between hepatic function and portal blood supply was initially based on surgical anastomosis of the portal vein to the caudal vena cava (Eck's fistula). Reports of naturally occurring portosystemic shunts are increasing due to a higher incidence of the syndrome and/or an increased awareness by veterinary practitioners. A case report of congenital portosystemic shunting is presented here with a discussion of possible pathogenesis, radiography, clinical pathology and treatment regimens.

CASE REPORT

On June 2, 1982, a 5 lb., 2½-year-old female Yorkshire terrier was referred to the ISU Veterinary Teaching Hospital. Presenting problems included pacing, restlessness and apparent blindness. The dog had been vaccinated for distemper, parvovirus, leptospirosis, canine adenovirus II and rabies.

Between October 1980 and March 1981 the dog had several episodes of tonsillitis accompanied by vomiting, occasional diarrhea and occasional itching. A tonsillectomy was performed March 19, 1981. From August 1981 to January 1982 there were further episodes of vomiting, diarrhea and itching. The condition seemed to respond to antibiotics and steroids.

On April 13, 1982 the dog had a bout of vomiting, and diarrhea with only partial response to therapy. The animal was restless and continuously paced the floor. Vomiting continued with gagging and anorexia. No significant abnormalities were seen radiographically.

On presentation to ISU the dog was restless, had eaten some food but exhibited no vomiting, and had feces of normal consistency. The dog had lost two pounds over a period of several weeks and appeared emaciated. Slight proprioceptive deficits were apparent in all four limbs. The dog was hospitalized and blood and urine samples were taken for analysis. Significant abnormalities included hypocalcemia (8.7 mg/dl), hypoalbuminemia (2.8 g/dl), low normal BUN (11 mg/dl) and slightly increased SGPT (79.3 IU/L). There was also a relative polycythemia and decreased numbers of platelets. Urinalysis showed a triple phosphate crystalluria.

An ammonia tolerance test was performed on the following day. An abnormal increase in venous blood ammonia was noted after administration of ammonium chloride orally. Fasting venous blood ammonia was 150 ug/dl while 30 minutes post-administration the level was 241 ug/dl. A tentative diagnosis of hepatic insufficiency was made based on these findings. A portosystemic shunt was suspected.

On June 4, 1982 the dog was sent home on a restricted protein diet of rice and cottage cheese. The owner was informed of the poor prognosis with medical management alone.

On June 8, the dog was readmitted to the

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clinic for operative portal venography. Anesthesia was induced with methoxyflurane administered via face mask. A ventral midline laparotomy was performed and a jejunal mesenteric vein was catheterized using a 22 gauge intravenous catheter. Survey radiographs were taken and then 2 cc of contrast medium[a] was hand-injected. Four lateral projection radiographs were taken at 0.5 second intervals. A single ventrodorsal (V-D) view was taken following a second injection of 2 cc of contrast medium.

Both lateral and V-D projections showed a large central venous shunt from the origin of the portal vein to the caudal vena cava in a post-hepatic location. The shunt was larger in diameter than the cranial portion of the portal vein in the lateral view. There was no evidence of opacification of the normal portal vein and its hepatic branches within the liver.

Following return to surgery and exploration of the craniodorsal abdomen, the shunt vessel could be seen extending dorsally from the portal vein to the caudal vena cava, cranial to the renal veins. The shunt was temporarily obstructed and observation of the intestinal serosa and mesenteric veins for 10 minutes revealed no signs of congestion. Radiography was performed following complete occlusion of the shunt. Lateral and V-D views showed filling of the remainder of the portal vein with evidence of branching of vessels into the hepatic parenchyma. The V-D view showed well-defined vessels in the area of the right medial and lateral lobes as well as the caudate lobe of the liver. Vessels supplying the left medial and lateral liver lobes were poorly opacified and lacked arborization.

Complete ligation of the shunt was performed, the mesenteric catheter was removed and the area of the venipuncture ligated. Routine closure of the abdomen was performed and the dog was allowed to recover from anesthesia under close observation. She was sent home June 10 with a steadily improving appetite. The owners reported problems with vomiting late at night but this was alleviated when the feeding schedule was changed to feeding throughout the day and night. At last report the dog had gained about 2 lbs. and was bright, alert and energetic.

[a]Hypaque-M75[R], Winthrop Lab., New York, New York.

DISCUSSION

Five major types of naturally occurring portosystemic anomalies have been recognized: (1) patent ductus venosus, (2) atresia of the portal vein with development of accessory portosystemic shunts, (3) anomalous connection of the portal vein to the caudal vena cava, (4) anomalous connection of the portal vein to the azygous vein, and (5) anomalous connection of the portal vein and caudal vena cava to the azygous vein.[4]

CLINICAL SIGNS

Clinical signs associated with portosystemic shunts are related to hepatic insufficiency due to altered composition of blood flow to the liver. Neurologic abnormalities are common and include listlessness, depression, compulsive pacing or circling, head pressing, ataxia, seizures, sudden viciousness, staring, blindness, hypermetria, tremors, coma or stupor. Stunted growth, weight loss and gastrointestinal disorders including vomiting, diarrhea, hypersalivation and anorexia can be seen. Ascites and polyuria/polydipsia have also been reported.

CLINICOPATHOLOGIC ABNORMALITIES

Blood analysis usually reveals slight to moderate elevations of SGPT and SAP, increased numbers of leukocytes (usually due to neutrophilia), low cholesterol, increased serum bilirubin and hypoproteinemia.

Anemia, commonly seen in Eck's Fistula dogs,[4] has not been associated with congenital portosystemic shunts in most cases, although Griffiths, et al. did find a significant correlation between erythrocyte microcytosis and portosystemic shunts, which may be related to anemia in some dogs.[5]

Hypokalemia can often be found especially in cases exhibiting hepatic encephalopathy.[6,7] Blood urea nitrogen levels are usually low to below normal.[8]

Urinalysis will often lead to the detection of ammonium biurate crystals. Increased serum ammonia and concentration of the urine contribute to this significant finding.[6] Some reports of urate crystalluria have also been made.[9] These have not yet been found in portosystemic shunts in cats.

PATHOGENESIS

Hepatic function appears to be dependent on portal blood composition rather than total blood flow. Attempts to increase arterial blood supply in congenital shunt cases have not alleviated clinical signs.[10] An essential hepatotrophic portal blood factor has been reported. Research indicates this factor may be produced by the pancreas, involving glucagon and/or insulin.[11,12]

The neurologic abnormalities associated with hepatic insufficiency have been referred to as hepatic encephalopathy (HE). Portal vascular anomalies are just one of a number of disorders that can produce this syndrome.

Numerous toxins have been incriminated in the pathogenesis of HE including ammonia, mercaptans, short chain fatty acids, false neurotransmitters (octapamine, gamma-amino-butyric acid, beta-phenylethanolamines) and increased plasma concentrations of the aromatic amino acids phenylalanine, tyrosine and tryptophan. Some researchers feel that the syndrome is actually a combination of several of these factors.[10,13,14]

Much emphasis has been placed on the role of ammonia in HE. Gastrointestinal (GI) production of ammonia is a primary source. Many bacterial species that reside in the GI tract are capable of hydrolyzing urea by way of the enzyme urease. This enzyme has been determined to be exclusively of bacterial origin.[7] Bacterial metabolism of ingested protein and products of protein digestion or urea, produce 40 percent of the GI ammonia while the remainder comes from non-bacterial sources including dietary ammonia and protein. Additional ammonia comes from the kidney, liver, muscle, nerve tissue, brain and erythrocytes.[15]

Ammonia is removed from the blood and tissues by intrahepatic urea formation and extrahepatic amination of ketoacids.[16] Ammonia uptake by the liver is related to the blood flow. Aldrete, et al. determined that 13 to 19 percent of the ammonia reaching the liver of dogs with experimental portacaval transposition bypassed metabolism to urea and re-entered the circulation as measurable ammonia.[16]

Movement of ammonia into and out of the vascular system is a very rapid process and conversion into glutamine in the body tissues can be completed within seconds. Body cells are freely permeable to ammonia but are almost impermeable to ammonium ion. Relative amounts of each are determined by pH (being in equilibrium, $NH_3 + H^+ \rightleftharpoons NH_4^+$). At the physiologic pH of 7.4 almost all of the ammonia of the blood is in the ionized form.[15] Metabolic alkalosis associated with hypokalemia will shift the equilibrium toward the more toxic, free NH_3.[6,7]

There are several theories to account for the toxicity of ammonia in the brain including interference with brain energy metabolism, accumulation of the inhibitory neurotransmitter GABA, a decrease in the neurotransmitter acetylcholine, a direct inhibitory effect on the neuronal membrane and a toxic intermediary metabolite.[17,18] Of these, interference with brain energy metabolism appears to warrant the most support.[14,18]

Inability to correlate the magnitude of hyperammonemia with the degree of encephalopathy has stimulated interest in a second important theory as to the cause of CNS dysfunction. Specific amino acid patterns have been found to be present in the blood of animals with HE.[13,17,19,20,21] The plasma concentrations of branched chain amino acids (BCAA) valine, leucine and isoleucine have been found to be lower than normal in dogs with portosystemic vascular anastomosis, whereas the aromatic amino acids (phenylalanine, tyrosine and tryptophan) are found in increased concentrations. In the normal dog the ratio of branched chain to aromatic amino acids is greater than 3 while with HE the ratio has been found to be about 1–1½.[13,17,19]

The low concentrations of BCAA are thought to be due to increased utilization by muscle and adipose tissue for energy.[5,19,22] There is controversy over the reason for the increased utilization. Some believe it is promoted by increased serum insulin stimulating uptake and catabolism of BCAA by muscle,[19] while others relate it to glucagon release associated with hyperammonemia (glucagon promoting gluconeogenesis from amino acids).[13,20] Hyperglucagonemia, hyperinsulinemia, hyperglycemia and decreased concentrations of BCAA have been seen in normal dogs to which ammonium chloride was administered.[20]

Impaired hepatic metabolism of the aromatic amino acids is the most likely cause of their increased plasma concentrations.

Branched chain and aromatic amino acids compete for transport into the brain. Decreased concentrations of BCAA would allow more of the aromatic amino acids to be transported. These may impair cerebral catechol-

amine metabolism, causing an increased synthesis of serotonin (tryptophan being a direct precursor of serotonin) and decreased synthesis of norepinephrine (due to excess phenylalanine which inhibits tyrosine-3-hydroxylase and allows tyrosine to be converted to octapamine, a false neurotransmitter).[13,19,20]

It is probable that the GI signs are also due to the accumulation of toxins in the brain, but this is unsupported as yet.

Ascites and edema are a result of the transudation of fluid associated with decreased plasma oncotic pressure due to hypoproteinemia. The liver is the sole source of plasma albumin and fibrinogen and hypoalbuminemia is a consistent finding in cases of portosystemic shunting.[6,8]

DIAGNOSIS

Specific testing of hepatic function can be an aid to evaluation of disease involving the liver. Three types of tests are useful: (1) those that measure serum enzyme activity, (2) those that measure bilirubin or organic dye secretion and excretion, and (3) those that measure a specific metabolic function such as synthesis of plasma proteins.[23]

The most important serum enzymes in hepatic disease are serum glutamic pyruvic transaminase (SGPT, recently referred to as alanine amino transferase) and serum alkaline phosphatase (SAP).[23] SGPT is liver specific in the dog and cat although small amounts do exist in kidney and heart muscle, so moderate increases can occur with severe renal or cardiac disease.[24] Increased SGPT is usually indicative of hepatocellular necrosis and inflammation. Slight to moderate elevations are seen in cases of portosystemic shunting.

SAP is actually a group of serum isoenzymes derived from several different body tissues.[25] The highest levels are found in the liver, kidney, bone, intestine and placenta, and increased serum levels are usually associated with obstruction of bile outflow and increased production. Portosystemic shunting produces slight to moderate elevations in SAP. Increased SAP levels can also result from bone abnormalities such as osteosarcoma or secondary hyperparathyroidism. It may also be a normal finding in growing dogs of large breeds.[23] SAP determinations are not useful in the cat as the liver concentration of SAP in cats is less than 25 percent of that in dogs.[23]

Bromsulphthalein (BSP) is an organic dye that is normally excreted in bile after conjugation with glutathione within hepatocytes.[3] Retention of dye is measured 30 or 45 minutes after I.V. injection. In normal dogs, BSP retention is less than five percent. Cases with portal venous anomalies generally have an increased retention.

BSP binds to albumin in the blood following injection. This may be important in assessing liver function in hypoproteinemic animals. Prolonged BSP retention due to decreased hepatic function may be offset by increased excretion due to decreased protein binding and may result in an apparently normal BSP clearance.[26,27]

The ammonia tolerance test (ATT) is a method of determining the liver's ability to convert ammonia to urea. In this test, an ammonium salt is administered orally after a 12 hour fast. Venous blood samples are taken prior to administration of the salt and again 30 minutes after administration. In normal dogs there is no significant increase in venous blood ammonia from the fasting state to 30 minutes post-administration. Dogs with portosystemic shunting will exhibit marked and significant rises in blood ammonia levels. Fasting venous blood ammonia levels alone may be greatly elevated. The ATT may be a more sensitive test of altered hepatic function than BSP retention, as cases have been found with normal BSP retention but with increased blood ammonia levels.[28] Cats may be more sensitive to oral ammonium chloride than dogs, as one cat exhibited signs of HE after administration.[29]

Abnormally low serum cholesterol levels have been found, presumably because the liver is the main source.[30] Blood urea nitrogen levels are usually low probably due to decreased urea cycle activity in the liver.[8]

RADIOGRAPHY

Survey radiographs may be helpful for a tentative diagnosis of congenital portal vein anomalies, although their usefulness may be limited by the lack of natural intra-abdominal contrast in young or emaciated dogs. A small liver is a common finding. Direct visualization of the liver may be impossible but size can be evaluated indirectly by locating gas within the lumen of the stomach and cranial duodenal flexure. Normally the stomach shadow lies parallel to the ribs in the lateral view, whereas, if the liver is small, the stomach will be more vertical in orientation, lying unusually close to

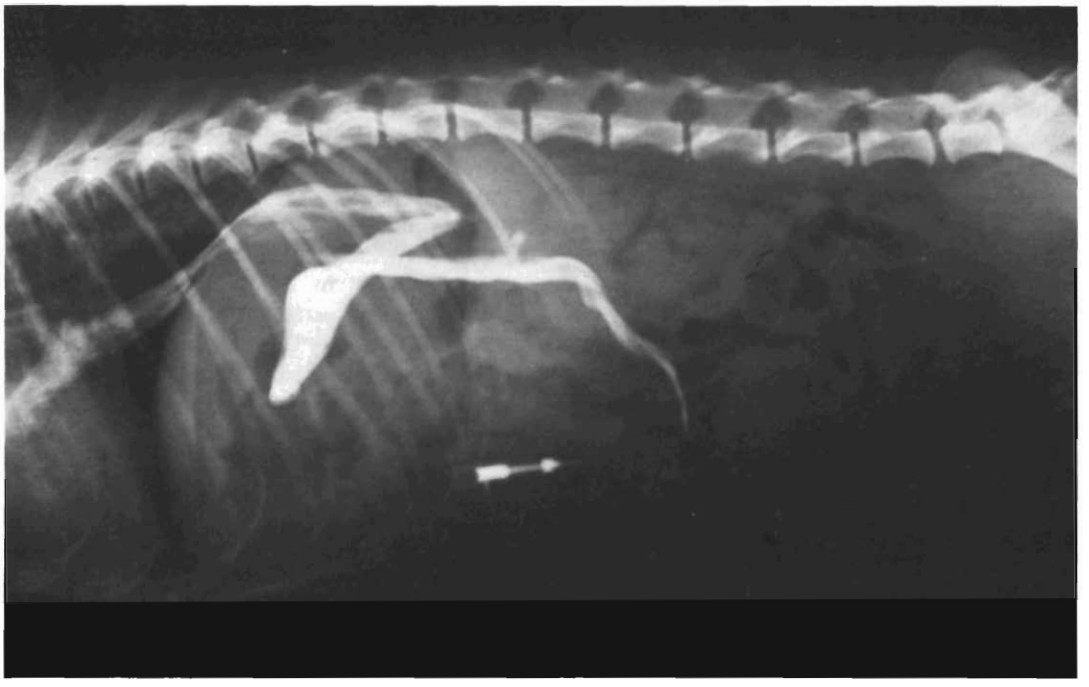

Fig. 1. Pre-ligation portal venogram

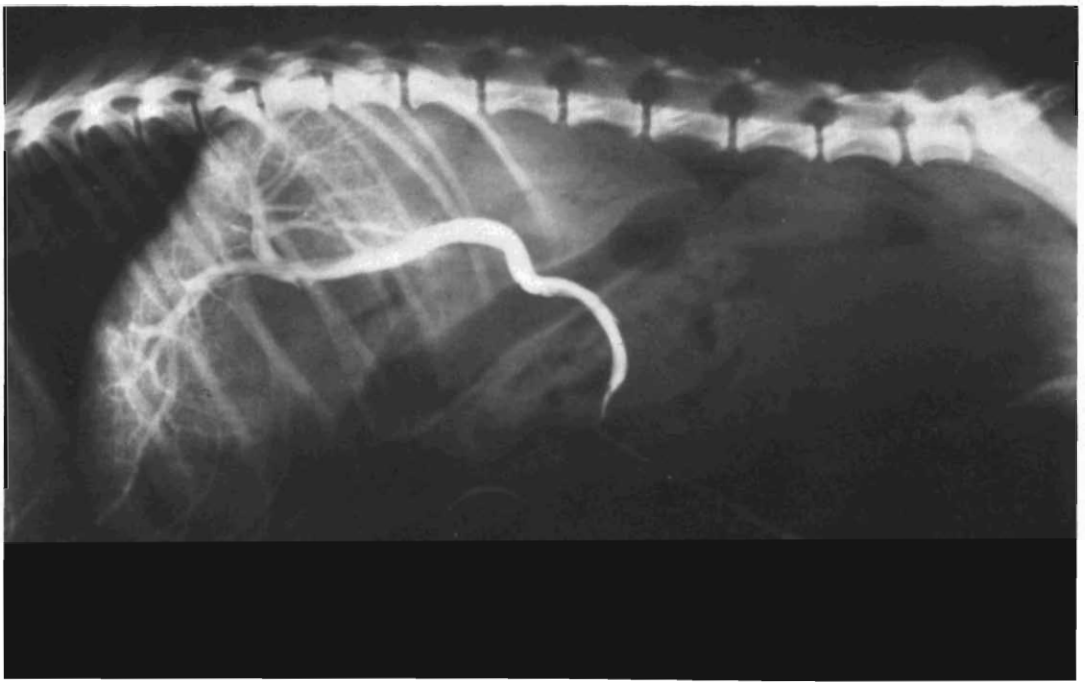

Fig. 2. Post-ligation portal venogram

the diaphragm.[4]

In cases where intra-abdominal contrast is poor, oral barium sulfate may be given to opacify the stomach and duodenum and thus outline the caudal border of the liver.

Renal enlargement has been reported in dogs with shunts.[4,30-32] The reason behind this is unknown.

Contrast radiography is extremely useful in the diagnosis of shunts. Cranial mesenteric arterial portography (CMAP), splenoportography, and portography have been found to be the most useful.

Selective catheterization of the visceral aortic branches, most commonly the cranial mesenteric artery permits the portal system to be outlined during the venous phase of an arterial injection of contrast medium.[31] This is performed under general anesthesia via a femoral artery cutdown, placing the catheter under fluoroscopic monitoring. Quality of CMAP may be inconsistent as it requires delivery of a large quantity of the contrast medium to the venous system as a bolus. Problems can occur if the contrast medium is injected too fast and under too much pressure, as regurgitation of the contrast into the abdominal aorta can occur and result in a poor quality study.[31] Catheter slippage from the artery (due to force of injection) can also occur leading to a non-selective aortogram. Delayed passage of contrast medium can lead to dilution and poor vascular contrast. It has been reported that improved quality may be seen if injections of vasodilators, such as prostaglandin E_1,[33] or epinephrine are given before administration of the contrast medium.[31]

The venous phase of arterial portography has been reported to be poor. A problem has been superimposition due to retention of contrast in the gastric, splenic or hepatic capillary beds thus preventing visualization of the sites of drainage of the gastrosplenic and gastroduodenal veins.

More consistent visualization of the portal system is obtained with splenoportography than with arterial portography. This technique is performed best under general anesthesia with the dog in right lateral recumbency. A catheter is passed obliquely into the splenic parenchyma via a small skin incision while the spleen is palpated externally. The catheter is positioned so as to lie near to the hilus of the spleen, thus allowing maximum venous drainage of contrast medium. Fluoroscopic monitoring is important for proper positioning.[34] Lack of experience with this technique can lead to accidental puncture of other abdominal organs, splenic laceration and hemorrhage or catheter slippage from the splenic parenchyma. Contrast medium seepage into the peritoneal cavity can interfere with maximal visualization of abdominal detail.[34] Shunts from the mesenteric venous system to the systemic circulation cannot be seen with this technique. In cats, splenoportography is best performed during laparotomy as the spleen can not be adequately localized by palpation.[34] The splenic vein can be catheterized during laparotomy in the dog also.

Operative mesenteric portography is a more invasive procedure than percutaneous angiography or splenography requiring general anesthesia and a laparotomy.[31,34] A catheter is placed in a mesenteric vein and sutured in place. The abdomen is temporarily closed for the contrast studies and then is re-opened to remove the catheter. The vein is ligated after the catheter is removed. An advantage to this procedure is that surgical ligation of the shunt can be performed while the abdomen is open, if contrast studies deem it to be feasible. Also, fluoroscopy is not required. The quality of this study is limited by the speed of injection of contrast, which is in turn limited by the size of catheter used. Dilution of the contrast is likely if it is injected too slowly. Ligation of the mesenteric vein can result in venous stasis and possible thrombosis.[34]

Other methods of contrast radiography have been developed which may be useful in diagnosing portal vein anomalies, but the ones mentioned here appear to be the most commonly used.

Contrast radiography has been used to help determine the feasibility of congenital portacaval shunt ligation by examining the circulation to the liver. The absence of portal vein branches entering the liver would prohibit any surgical correction. But several researchers have determined that the inability to visualize any portal parenchymal circulation may not be due to the absence of such circulation, but rather to the preferential flow of blood through the less resistant shunt.[29,35] Selective shunt occlusion should be performed to accurately assess hepatic perfusion.

EMERGENCY THERAPY

Animals that present with acute hepatic encephalopathy should be completely withdrawn from oral and parenteral protein sources. Intravenous fluids should be administered to correct dehydration and dilute toxins, correct alkalosis or hypokalemia and enhance renal elimination of toxins. Ringer's solution is preferred over lactated Ringer's due to the alkalinizing effect of the latter. Intravenous glucose and fructose may be useful as energy sources to prevent protein catabolism and the associated ammonia production.[3,6] Fluids are contraindicated in cases with ascites.

Management of ascites may involve the use of diuretics, however the patient must be observed for hypokalemia, alkalosis and hypovolemia when they are used.[22]

Blood transfusions are contraindicated and GI bleeding must be controlled as these can increase the hyperammonemia.[17,22]

Warm water enemas are indicated to clear the colon of nitrogenous products and urease-producing bacteria. Oral administration of neomycin or kanamycin, which are classified as gut sterilizers due to insignificant absorption, also help reduce enteric bacterial numbers.[24]

Encephalopathic effects may be reduced by oxygen therapy since the hepatoencephalopathic brain has been found to be more sensitive to hypoxia and electrolyte imbalances.[3] This will also correct any renal or hepatic hypoxia and decrease enterohepatic circulation of urea nitrogen.[17]

Convulsions appear to be most responsive to low dosages of diazepam (Valium) and chlordiazepoxide (Librium).[24]

Barbiturates and tranquilizers should be avoided due to impaired metabolism by the liver and increased sensitivity of the brain, thus potentiating their duration of action and toxicity.[4,21,30,36]

Intravenous antibiotics may be indicated for concurrent infections. Chloramphenicol or sodium penicillin (alone or in combination with kanamycin) have been recommended.[17]

If analgesia is required, reduced dosages of meperidine, codeine or acetaminophen are recommended over morphine.[24]

LONG-TERM MEDICAL MANAGEMENT

Although the usual course for management of shunts has been medical, the success of such therapy has been poor. Progressive deterioration due to hepatic fibrosis and atrophy has been the rule.[4,6]

Oral protein intake must be restricted. Small amounts of protein with high biologic value (percentage of protein absorbed and retained) should be fed frequently throughout the day. These will be maximally catabolized before they reach the large intestine thus preventing action by the urea-splitting bacteria. Proteins of animal origin are better than those of plant origin as the latter are often deficient in lysine, methionine, leucine and tryptophan.[37] Recommended proteins are eggs and cottage cheese. Fish meal, shellfish and animal gland products should be restricted as they contain purine and precursors of uric acid.[37]

Restoring plasma concentrations of BCAA may be important in treatment. Parenteral supplementation with specific mixture of synthetic amino acids has been used to correct imbalances.[13] Cottage cheese appears to provide an unidentified substance that has been found to aid cases of HE, besides being a rich source of BCAA.[19,38] However, its usefulness is limited as dogs don't readily accept it.

The carbohydrate source is also an important consideration. The diet should contain a high level of carbohydrates which are completely digestible within the small intestine. This is important as metabolism to volatile fatty acids (VFA) can occur within the colon, and VFA have been found to be synergistic with hyperammonemia in producing the signs of HE.[37] Starch and dextrose from rice and corn syrup are readily available sources of energy.

Energy sources must be correlated to protein intake to prevent energy deficits. In energy deficits, BCAA are utilized for energy, further depleting their plasma concentrations.[37]

Vitamin supplementation is warranted since the liver's ability to store and convert them to the biologically active compounds is impaired.

Long-term oral antibiotics have been recommended to decrease the colonic content of urea-splitting bacteria. Neomycin has been commonly used. However, because severe renal insufficiency may develop in the late stages of hepatic failure, treatment with neomycin must be carefully monitored because of ototoxicity or further injury to the kidneys, even with oral administration.

If ascites is a problem, a low sodium diet and judicious use of diuretics may be warranted. An oral potassium supplement may be

used to avoid hypokalemia.[17]

Lactulose (beta-1,4-galactoside fructose) is a non-absorbable synthetic disaccharide which has been used in cases of portal vein anomalies which are unresponsive to protein restriction and oral neomycin. It is given orally and is hydrolyzed in the colon to lactic, formic and acetic acids which produce an acid environment and shift the $NH_3 + H^+ \rightleftharpoons NH_4^+$ equilibrium away from ammonia. The acid environment favors the growth of *Lactobacillus* organisms which do not split urea.[17,29] Lactulose also acts as a cathartic, decreasing intestinal contact time and thus decreasing the amount of ammonia produced. Side effects associated with its use include anorexia, vomiting, diarrhea, abdominal pain and flatulence.[17]

Methionine has been used because of its lipotropic properties, however, caution is indicated as it is metabolized to mercaptans within the intestine. These may be synergistic with ammonia in producing hepatic coma.[3,7,22]

Anabolic steroids have been shown to decrease protein catabolism and improve liver function in man if administered over long periods of time. However, it is very important that adequate protein and energy be available for optimum action.[24]

L-dopa has been used in experimentally created portacaval shunt cases and may decrease blood ammonia. It may replace false neurotransmitters and thus be a protection against hepatic encephalopathy.[17]

SURGICAL TREATMENT

Surgical ligation of congenital portosystemic shunts has recently gained some support in the literature. If contrast radiography reveals hepatic circulation and one or a few shunt vessels, ligation may be feasible. Ligation of acquired shunts should not be attempted.

A significant determinant of the success of ligation in a patient is measurement of portal venous pressure. Normal portal venous pressure in the dog is 8–12 cm. H_2O.[29,30] Rapid marked increases in pressure after occlusion of the shunt indicate that subtotal ligation should be considered rather than complete ligation.

Temporary occlusion of the shunt over several minutes while observing the viscera for signs of venous congestion and engorgement can be useful if a manometer is not available for determining pressure. However, Breznock reported two cases of complete shunt ligation where stagnant hypoxia of the viscera was not noted until three to five hours after occlusion.[30]

Subtotal ligation of shunts in very young dogs may gradually lead to total occlusion as the relative size of the lumen decreases with age.[35] In older dogs a second procedure at a later date to further decrease the size of the lumen may be possible as hepatic portal circulation increases.

Strombeck, et al. attempted total surgical ligation of a shunt vessel in combination with arterialization of the liver by transposition of a section of jejunum, but this failed.[10]

Preparation of the patient for surgery necessitates careful use of drugs due to impaired liver function. Therefore a good anesthetic protocol would include narcotic premedication (reversible with naloxone) and mask induction preferably with a mixture of methoxyflurane, nitrous oxide and oxygen. Halothane has been found to be hepatotoxic in combination with hypoxia and metabolic acidosis.[24]

Consideration of hepatic dysfunctions would lend support to glucose administration during surgery and possibly administration of plasma proteins before anesthesia, if serum levels of albumin are very low.

SUMMARY

The involvement of the liver in maintaining homeostasis in multiple body systems makes diagnosis and treatment of liver disease imperative for life. A wide variety of clinical signs can occur with liver disease. Many tests of hepatic function are available to the veterinarian to confirm or supplement clinical impressions. This suggests that no single test is sufficient for diagnosis of a liver disorder. A simple screening panel (i.e., complete blood count, BUN, SGPT, SAP, Albumin, Cholesterol, Bilirubin and Glucose) should be done initially, and tests subsequent to this should build upon its results. The ATT and BSP retention have proven very useful. Contrast radiography is called for only after sufficient evidence of shunting has been gathered. Of the confirmatory radiographic techniques, arterial portography or splenoportography are less invasive and appear to be sufficient to identify shunts provided the necessary equipment is available. However, if this is not the case, operative mesenteric portography is indicated. If surgical ligation of the shunt is to be attempted, then operative portography is definitely indicated.

Medical management only slows the progressive liver atrophy and fibrosis and ame-

liorates the clinical signs. If feasible, surgical ligation of the shunt should be performed to maximize hepatic function and allow a more normal life.

REFERENCES

1. Turnwald GH: Portal azygous anomaly and hepatic encephalopathy in a dog. *Austr Vet Pract* 9:5–9, 1979.
2. Bradley SE: The Hepatic Circulation, in Hamilton and Dow (ed.): *Handbook of Physiology Section 2: Circulation Vol II.* American Physiological Society, Washington, DC, pp 1387–1438, 1963.
3. Rubin GJ, Jones BD: The Liver, in Bojrab MJ (ed): *Pathophysiology in small animal surgery.* Lea and Febiger, Philadelphia, PA, pp 129–141, 1981.
4. Ewing GO, Suter PF, Bailey CS: Hepatic Insufficiency Associated with Congenital Anomalies of the Portal Vein in Dogs. *JAAHA* 10:463–476, 1974.
5. Griffiths GL, Lumsden JH, Valli VEO: Hematologic and Biochemical Changes in Dogs with Portosystemic Shunts. *JAAHA* 17:705–710, 1981.
6. Cornelius LM, et al.: Anomalous Portosystemic Anastomoses Associated with Chronic Hepatic Insufficiency in Six Young Dogs. *JAVMA* 167:220–228, 1975.
7. Tennant BC, Hornbuckle WE: Diseases of the Liver, in Anderson NV (ed): *Veterinary Gastroenterology.* Lea and Febiger, Philadelphia, PA, pp 593–620, 1980.
8. Campbell TM, Lording PM, Wrigley RH, Lavelle RB: Portal Vein Anomaly and Hepatic Encephalopathy in Three Dogs. *Austr Vet J* 56:593–598, 1980.
9. Barrett RE, Delahunta A, Roenick WJ, Hoffer RE, Coons FH: Four Cases of congenital portacaval shunt in the dog. *J Sm Ani Pract* 17:71–85, 1976.
10. Strombeck DR, Breznock EM, McNeel S: Surgical Treatment for Portosystemic Shunts in Two Dogs. *JAVMA* 170:1317–1319, 1977.
11. Whittemore AD, et al.: Hepatic Regeneration in the absence of portal viscera. *Surgery* 77:419, 1975.
12. Duguay LR, Lee S, Orloff MJ: Regulation of Liver Regeneration by the Pancreas. *Gastroenterology* 70:980, 1976.
13. Fischer JE, et al.: The role of plasma amino acids in hepatic encephalopathy. *Surgery* 78:276–290, 1975.
14. Schenker S, Breen KJ, Hoyumpa AW JR: Hepatic Encephalopathy: Current Status. *Gastroenterology* 66:121–151, 1974.
15. Schalm SW, DeGroot GH: Hepatic Encephalopahy, in Arias IM, Frenkel M, Wilson JHP (ed): *The Liver Annual 1 1981,* Exerpta Medica, Amsterdam-Oxford-Princeton, pp 166–182, 1981.
16. Aldrete JS: Quantification of the Capacity of the Liver to Remove Ammonia From the Circulation of Dogs with Portacaval Transposition. *Surg Gyn Obstet* 141:399–404, 1975.
17. Barrett RE: Canine Hepatic Encephalopathy, in Kirk RW (ed): *Current Veterinary Therapy VII Small Animal Practice,* WB Saunders Co, Philadelphia, PA, pp 822–829, 1980.
18. Strombeck DR, Weiser MG, Kaneko JJ: Hyperammonemia and Hepatic Encephalopathy in the Dog. *JAVMA* 166:1105–1108, 1975.
19. Strombeck DR, Rogers Q: Plasma Amino Acid Concentrations in Dogs with Hepatic Disease. *JAVMA* 173:93–96, 1978.
20. James JH, et al.: Hyperammonemia, Plasma amino acid imbalance and Blood: Brain amino acid transport: A Unfied Theory of Portal-Systemic Encephalopathy. *Lancet* 2:772–775, Oct 13, 1979.
21. Strombeck DR, Rogers Q, Stern JS: Effects of Intravenous Ammonia Infusion on Plasma Levels of Amino Acids, Glucagon and Insulin in Dogs. *Gastroenterology* 74:1165, 1978.
22. Sherding RG: Hepatic Encephalopathy in the Dog. *Comp. Cont Educ 1:* 55–63, 1979.
23. Goldston RT, Wilkes RD, Seybold IM: The Clinical Pathology Laboratory Hepatic Function Test—1: Hepatic Enzymology. *VM/SAC* 76:642–645, 1981.
24. Hardy RM: Diseases of the Liver, in Ettinger SJ (ed): *Textbook of Veterinary Internal Medicine,* WB Saunders Co, Philadelphia, PA, pp 1219–1246, 1975.
25. Kidder DE, McCullagh KG: Hepatic Function and Liver Function Tests, in Phillipson AT, Hall LW, Pritchard WR (ed): *Scientific Foundations of Veterinary Medicine,* William Heinemann Medical Books LTD, London, pp 277–285, 1980.
26. Duncan JR, Prasse KW: *Veterinary Laboratory Medicine. Clinical Pathology,* ISU Press, Ames, IA, pp 79–94, 1977.
27. Goldston RT, Wilkes RD, Seybold IM: The Clinical Pathology Laboratory. Hepatic Function Test—2: Bilirubin metabolism, bromsulphthalein (BSP) excretion test. *VM/SAC* 76:795–797, 1981.
28. Meyer DJ, et al.: Ammonia Tolerance Test in Clinically Normal Dogs and in Dogs with Portosystemic Shunts. *JAVMA* 173:377–379, 1978.
29. Levesque DC, et al.: Congenital portacaval shunts in two cats: Diagnosis and surgical correction. *JAVMA* 181:143–145, 1982.
30. Breznock EM: Surgical Manipulation of Portosystemic shunts in Dogs. *JAVMA* 174:819–826, 1979.
31. Suter PF: Portal Vein Anomalies in the Dog: Their Angiographic Diagnosis. *J Am Vet Rad Soc* 16:84–97, 1975.
32. Audell L, Jonsson L, Lannek B: Congenital Portacaval Shunts in the Dog. A Description of Three Cases. *Zbl Vet Med A,* 21:797–805, 1974.
33. Hornof WJ, Suter PF: The use of prostaglandin E_1 and tolazoline to improve cranial mesenteric arterial portography in the dog. *J Am Vet Rad Soc* 20:15–21, 1979.
34. Schmidt S, Suter PF: Angiography of the Hepatic and Portal Venous System in the Dog and Cat: An Investigative Method, *Vet Rad* 21:57–77, 1980.
35. Gofton N: Surgical Ligation of Congenital Portosystemic Venous Shunts in the Dog: A Report of Three Cases. *JAAHA* 14:728–733, 1978.
36. Lohse CL, Selcer RR, Suter PF: Hepatic Encephalopathy Associated with Situs Inversus of Abdominal Organs and Vascular Anomalies in a Dog. *JAVMA* 168:681–688, 1976.
37. Fox SM, Sturtevant FC: Dietary Management of a dog with Hepatic Insufficiency suggestive of a portasystemic shunt. *VM/SAC* 75:1384–1391, 1980.
38. Patel D, Anderson GH, Jeejeebhoy KN: Amino Acid Adequacy of Parenteral Casein Hydrolysate and Oral Cottage Cheese in patients with Gastrointestinal Disease as measured by Nitrogen Balance and Blood Aminogram. *Gastroenterology* 65:427–437, 1973.